

# rCBF SPECT in patients after mild cranio-cerebral trauma

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## Abstract

**BACKGROUND:** The aim of the study was to compare SPECT and CT scanning results in patients after mild head trauma, the former in the aspect of both focal and regional perfusion deficits. **METHODS:** High-resolution <sup>99m</sup>Tc-HMPAO SPECT was performed on a group of 31 patients after mild head trauma using a three-head gammacamera, qualitatively and quantitatively, utilising an asymmetry index for unilateral perfusion deficits and a comparison to cerebellar perfusion for assessing the regional cerebral perfusion. For assessing the normal values a control group of 30 patients was studied.

**RESULTS:** 27 studies were abnormal, 4 normal. Abnormalities consisted of focal perfusion deficits in 27 patients and diffuse regional hypoperfusion in 11 patients. Focal perfusion deficits were localised most frequently in frontal lobes and occipito-parietal area. Analysing individual cases, diffuse regional hypoperfusion was most pronounced in frontal lobes, analysing whole group a significant decrease of regional cerebral perfusion was found in all brain regions: frontal, occipital, parietal lobes, basal ganglia and thalami, except temporal lobes. A separate entity presents 4 patients in whom careful investigation revealed simulation of head trauma. In those cases perfusion brain SPECT was normal. CT scanning showed abnormalities in brain parenchyma in 11 patients, in bone in 9 patients. 11 patients showed persistent neurological symptoms and signs. Correlation between SPECT results and those signs was high in 7 of them, partial in 3, no correlation was seen in 2. **CONCLUSIONS:** Brain perfusion SPECT is a useful tool in the assessing of patients after mild head trauma. It shows higher sensitivity than brain CT, although it may lack specificity in some cases. SPECT shows high utility in discriminating cases of simulation. It may be a useful tool in forensic medicine.

**Key words:** cranio-cerebral trauma, cerebral blood flow, single photon emission computed tomography

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## Introduction

Central-nervous system (CNS) injury is the second most frequent complication of trauma, after limb lesions. CNS injury is frequent, especially in patients with multi-organ trauma and is often decisive about the outcome (1). About 1/3 of patients develop a post-concussion syndrome and 1/2 of these become unable to go back to work (2, 3).

There is a controversy as to whether those patients truly have an organic syndrome or are reacting on a basis of psychogenic and environmental factors (4). Clinically, light post-cranio-cerebral trauma is followed mostly by functional disturbances, which is met by the lack of diagnostic imaging techniques which enable the clinician to assess and follow-up those lesions. The objective clinical assessment, supported by adequate diagnostic imaging is important, facing the high incidence of decrease in professional activity and the legal claims by those patients. Especially the legal consequences of a head trauma demand an objective assessment, as most of head trauma is a result of either traffic accidents or criminal incidents.

At present there is no commonly acknowledged „golden standard” of brain diagnostic imaging in cranio-cerebral trauma, enabling the clinician to perform an objective assessment of brain disorder and an equally objective follow-up. Computed tomography is frequently performed, but usually reveals no abnormalities, the same with MRI scanning (5–10).

The aim of the study was to compare SPECT and CT scanning results in patients after mild head trauma, the former in the aspect of both focal and regional perfusion deficits.

## Materials and methods

### Subjects

The study involved 31 patients 2–14 days after cranio-cerebral trauma: 6 women and 25 men, mean age 38±15 years, range 18–75 years. As healthy controls there were 30 volunteers, 17 women, 13 men, age 42±13 years, range 20–74 years. Arterial hypertension, renal insufficiency, congestive heart disease, chronic alcoholism and medication with the possible influence on cerebral perfusion were the exclusion criteria. All patients gave informed consent. The project had the approval of the Local Medical Ethics Committee.

In all patients (n=31) brain concussion had been diagnosed. It was secondarily uncomplicated in 20 patients, in 11 patients simultaneously cerebral contusion, cerebral haematomas, regional

and/or generalised brain oedema, skull fracture and superficial wounds were seen. Autonomic disorders were seen in 24 patients, such as circulatory and/or respiratory instability (bradycardia, tachycardia, bradypnoe, tachypnoe, instability of blood pressure), nausea, vomiting, vertigo, headache.

Consciousness was assessed utilising Glasgow Coma Scale (GCS). GCS value was between 9–15 pts, mean GCS value was  $14.4 \pm 1.3$  pts.

In 13 patients (41.9%) head trauma followed slipping on uneven ground, in 10 (32.3%) it followed a car accident, in 8 (25.8%) a criminal incident. The mean time spent in hospital was  $15 \pm 9.3$  days.

### Procedure

Brain SPECT studies were performed in the first two weeks after the trauma, mean  $8.8 \pm 4$  days. Scanning was performed approximately 1 h following the intravenous injection of 740 MBq (20.0 mCi) of  $^{99m}\text{Tc}$ -HMPAO (Amersham, Amersham, UK). Scanning was performed on a triple-head gammacamera Multispect-3 (Siemens, Erlangen, Germany) using a low-energy, ultra-high resolution collimator.

The data were collected into a  $128 \times 128$  matrix, 4.8 mm per pixel. The raw data were smoothed with a Butterworth filter, cut-off frequency 0.35. Chang attenuation correction was not performed. The images were reoriented in the axial, coronal and sagittal planes. The data were displayed on a 10-grade colour scale. Focal perfusion abnormalities were read twice by two independent observers. Their depth was assessed utilising an asymmetry index (AI):  $\text{AI} = \frac{R - L}{(R + L)/2} \times 100\%$ , where R and L are mean counts/pixel values in the right and left hemisphere, respectively (11).

Regional cerebral blood flow was assessed semiquantitatively by calculating the index of regional mean counts/pixel values divided by those in the cerebellum.

As significant were considered focal perfusion deficits with asymmetry index values exceeding 2 standard deviations (SD) below the mean for the control group. As significant were considered regional cerebral blood flow deficits with regional/cerebellar ratio values 2 SDs below the mean in the control group (12). Statistic analysis was performed utilising Kruskal-Wallis test and non-parametric Spearman's test. A  $P$ -value  $< 0.05$  was considered significant.

### Results

The results of control group brain perfusion SPECT scanning are shown in Table 1.

**Table 1. Inter- and intra-hemispherical differences of  $^{99m}\text{Tc}$ -HMPAO uptake in the control group. Values are expressed as the percentage of tracer uptake**

Asymmetry index (AI) (mean $\pm$ SD)		Regional uptake/cerebellar uptake (mean $\pm$ SD)	
		Right hemisphere	Left hemisphere
Frontal lobe	$2.6 \pm 2.0$	$93.6 \pm 3.5$	$92.9 \pm 3.8$
Temporal lobe	$3.0 \pm 1.7$	$93.8 \pm 4.4$	$93.1 \pm 4.8$
Occipital lobe	$3.7 \pm 2.4$	$95.4 \pm 6.5$	$95.6 \pm 6.6$
Parietal lobe	$3.8 \pm 2.5$	$96.3 \pm 7.6$	$96.4 \pm 7.0$
Basal ganglia	$3.5 \pm 2.8$	$87.6 \pm 5.1$	$85.5 \pm 5.4$
Thalami	$4.6 \pm 2.5$	$88.7 \pm 7.5$	$85.4 \pm 7.3$

### Patients after head trauma

27 studies were abnormal, 4 were normal. Abnormalities consisted of focal perfusion deficits in 27 studies (87.1%) and diffuse regional hypoperfusion in 11 studies.

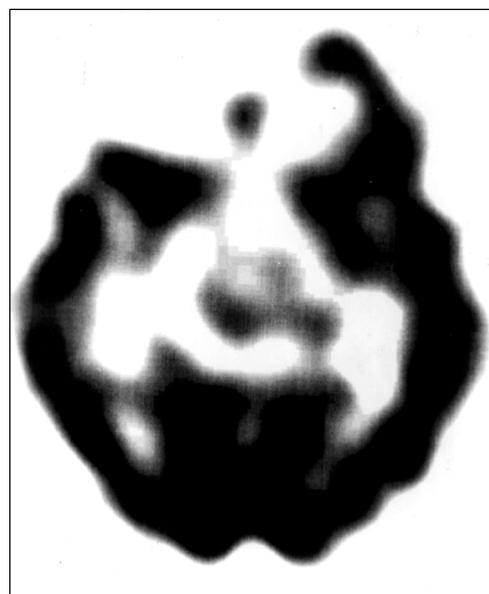
### Focal perfusion deficits

Mean number of focal perfusion deficits varied between 0–8 per patient, mean  $3.4 \pm 2.4$  per patient. The total number of focal perfusion deficits was 65. The mean number of focal perfusion deficits per patient varied in women —  $1.8 \pm 2.2$  ( $n=6$ ) and men —  $3.8 \pm 2.4$  ( $n=25$ ).

Focal perfusion deficits were localised mostly in frontal lobes — 18, temporal lobes — 12, occipital lobes and occipito-parietal border — 18, hippocampus — 8, thalamus — 2, basal ganglia — 3. Examples of focal perfusion deficits are shown in Figures 1 and 2.



**Figure 1.** Bifocal perfusion defect in occipito-parietal border after whiplash injury.



**Figure 2.** Focal perfusion deficit in right frontal lobe.

Mean asymmetry index for the focal perfusion deficits in frontal lobes was  $20.6 \pm 14.9\%$ , temporal lobes —  $18.2 \pm 7.6\%$ , parietal lobes —  $17.6 \pm 17.2\%$ , occipital lobes —  $15.3 \pm 6\%$ , hippocampus —  $16.2 \pm 6.1\%$ .

#### **Focal perfusion deficits and the mechanism of trauma**

Focal perfusion deficits were localised mostly in a site of trauma: in 22/27 of patients (81.5%). Some were accompanied with ones in a contralateral hemisphere in an axis of trauma (*contre-coup*): 7/27 of patients (25.9%), many were multifocal. As a site of trauma was considered an X-ray-confirmed site of fracture and/or a site of skin wound or concussion. The localisation of perfusion deficits depending on trauma site is shown in Table 2.

**Table 2. Localisation of local perfusion deficits**

Localisation of focal perfusion deficits	No of patients n=27 [%]
In trauma site	2 (7.4)
In trauma site and contralaterally	1 (3.7)
Multifocal, including trauma site	14 (51.9)
Multifocal, including trauma site and contralaterally	4 (14.8)
Multifocal, not linked with trauma site	4 (14.8)
Multifocal, including contralateral site	2 (7.4)

Analysing the relation between *contre-coup* localisation of blood flow changes and the mechanism of trauma, *contre-coup* pattern was found in 1/7 (14.2%) of patients after acceleration/deceleration type-trauma and in 9/24 (37.5%) of patients after acceleration-type-trauma.

#### **Focal perfusion deficits and GCS score**

All patients with GCS lower than 15 had at least 2 focal perfusion deficits. All patients with the number of focal perfusion deficits exceeding 6 were with GCS below 15. All patients with the lowest GCS index had the maximal number of focal perfusion deficits — 8.

There was no dependence between the number of the focal perfusion deficits per patient and the time between the trauma and introduction of treatment. All patients with the number of focal perfusion deficits larger than 6 were hospitalised during the first 24 h post-trauma. Also there was no correlation between the number of focal perfusion deficits per patient and the age of the patient.

A positive correlation was found between the number of focal perfusion deficits and the time between the trauma and performing brain SPECT scanning.

#### **Regional cerebral perfusion**

Diffuse hypoperfusion of the frontal lobes (hypofrontality) exceeding 2 SDs below the mean value of the control group was found in 11 patients.

In 10/11 patients with hypofrontality this was accompanied by the focal perfusion deficits. In patients with hypofrontality the mean number of focal perfusion deficits was  $5.5 \pm 2.1$ , in patients without hypofrontality  $2.3 \pm 1.8$  ( $p < 0.05$ ). There was no significant dependence between the time spent in hospital and the incidence of hypofrontality. Patients with hypofrontality stayed in hospital a mean  $19.2 \pm 11.5$  days, without it  $12.8 \pm 7.2$  days.

Regional perfusion of the remaining brain areas in patients after trauma did not exceed 2 SDs below the mean value of the control group, but on analysing this group as a whole statistically significant lower regional perfusion rates were found in frontal lobes, occipital lobes, occipito-temporal border, left parietal lobes, right hippocampus, thalami and basal ganglia ( $p < 0.05$ ). There was no difference in regional cerebral blood flow, as compared to the control group, in temporal lobes. The values of region/cerebellar indices for the particular regions are shown in Table 3.

#### **Brain SPECT and the clinical data**

Following trauma, persistent neurological symptoms and signs were found in 12/31 patients (38.7%). In 7 patients paresis was found, in 5 nystagmus, in 4 central-origin hearing impairment, in 3 meningismus, in 3 blurred vision, in 3 motor aphasia, in 2 hypoaesthesia, in 1 cerebellar ataxia.

All patients with neurological symptoms and signs had (with one exception) multifocal perfusion defects, 3–8 perfusion deficits per patient, mean 5 deficits.

In 7 patients focal perfusion deficits correlated with all neurological symptoms and signs. In 3 patients they correlated partially. In 2 patients no perfusion deficits corresponding to neurological symptoms and signs were found.

A separate entity presented a group of 4 young patients with uncomplicated brain concussion, without neurological symptoms and signs and normal SPECT scanning results. A thorough collecting of medical, psychological and sociological data aroused a high suspicion of brain concussion simulation in order to avoid army conscription as the reason for admission to the hospital. In this subgroup both qualitative and quantitative parameters of brain perfusion SPECT scanning did not differ significantly as compared with the control group.

#### **Brain SPECT results as compared to CT scanning**

Altogether 30 CT scans were performed on 20 patients. 8 patients had CT scanning performed two or three times.

In 9 patients CT scanning results were normal. In 11 patients the following changes were seen: regional cerebral oedema in 4 patients, generalised cerebral oedema in 1 patient, cerebral contusion in 3, paracerebral haematoma in 4, subarachnoid haemorrhage in 2. Bone lesion was seen in 9 patients: skull fracture in 4 patients, skull and skull's base fracture in 5 patients. In 3 patients diagnosis of skull fracture was based on the X-ray alone. In SPECT scanning 58 focal perfusion deficits had no equivalent in

**Table 3. Regional  $^{99m}\text{Tc}$ -HMPAO uptake compared to cerebellar uptake in control group and mild head trauma**

Brain region	Control group (n=30)		Mild head trauma (n=31)	
	Right hemisphere	Left hemisphere	Right hemisphere	Left hemisphere
Frontal lobe	$93.6 \pm 3.5$	$92.9 \pm 3.5$	$85.5 \pm 10.7^*$	$81.8 \pm 14.9^*$
Temporal lobe	$77.7 \pm 10.9$	$77.3 \pm 10.8$	$81.1 \pm 11.4$	$75.4 \pm 11.9$
Occipital lobe	$95.4 \pm 6.5$	$95.6 \pm 6.6$	$87.1 \pm 12.8^*$	$87.2 \pm 11.6^*$
Parietal lobe	$96.3 \pm 7.6$	$96.4 \pm 7.0$	$89.9 \pm 9.2$	$85.9 \pm 16.5^*$
Basal ganglia	$87.6 \pm 5.1$	$85.5 \pm 5.4$	$78.8 \pm 9.6^*$	$77.6 \pm 7.2^*$
Thalami	$88.7 \pm 7.5$	$85.4 \pm 7.3$	$76.8 \pm 8.4^*$	$75.9 \pm 8.9^*$

\* statistically significant

CT scanning. There was a concordance in SPECT/CT scanning findings in 14 cases. In CT only and not SPECT scanning 2 post-traumatic changes — paracerebral haematomas — were found.

## Discussion

There is increasing evidence that brain perfusion SPECT is superior to CT in detecting brain perfusion abnormalities (2, 4–10). Some data are given in Table 4.

**Table 4. Reference data on SPECT scanning in head trauma**

SPECT	CT	MRI	Author	Remarks
90%	72%	–	Gray, 1992	severe cranio-cerebral trauma
60%	25%	–	Masdeu, 1993	mild cranio-cerebral trauma
100%	0%	100%	Prayer, 1993	remote period after severe cranio-cerebral trauma
66%	34%	45%	Ichise, 1994	remote period after mild cranio-cerebral trauma
78%	40%	46%	Sataloff, 1996	neurootologic complications of cranio-cerebral trauma
90%	36%	–	Lass, 1996	remote period after cranio-cerebral trauma
59%	0%	–	Abu-Judeh, 1999	mild cranio-cerebral trauma with negative CT result

This study supports those data and should be discussed both in the aspect of its neuropathophysiological findings, as well as in the practical application of brain perfusion SPECT scanning in patients after cranio-cerebral trauma.

Our study suggests that brain perfusion abnormalities after cranio-cerebral trauma present as a mixture of focal perfusion deficits and diffuse regional perfusion alterations, involving most of the brain regions, excluding temporal lobes.

Focal brain perfusion deficits were shown mostly in frontal lobes, which is not surprising considering the fact that the frontal lobes present ca. 50% of brain volume and are localised in the area of the head frequently exposed to trauma. An equal number of focal brain perfusion deficits was found in occipital lobes with particular attention to occipito-parietal border. This area, lying on the border of the vascular areas of three main cerebral arteries, is particularly susceptible to cerebral ischaemia due to poor compensation *via* meningeal arteries (13).

The mechanism of the development of the focal perfusion deficit should in theory be clear: the direct impact of trauma at its site and eventually in the contralateral hemisphere (*contre-coup mechanism*), in a part of the patients followed by the sequelae of extracranial vessels lesion. This is eventually followed by the sequelae of early and late arterial vasospasm and repair processes.

In our material 4/5 of focal perfusion deficits were localised in the site of trauma, which could be expected, and in 1/4 on the axis of trauma in the contralateral hemisphere (probable *contre-coup* mechanism). We found a discrepancy with data from the area of forensic medicine, which attribute the *contre-coup* mechanism to the acceleration/deceleration mechanism, for example in car accidents (15, 16). Single contusion foci are met in acceleration mechanism (slipping or criminal attack) (16). Our data on CBF alteration are quite contrary: bifocal lesions were found more

frequently in patients with the latter mechanism of trauma. Such a phenomenon can be met in heavy acceleration-type trauma (17).

In the majority of patients focal or bifocal pattern of brain perfusion deficits has been accompanied by perfusion defects in other areas of the brain. This probably reflects either an impact of forces lying not directly on the axis of trauma (flexion and rotation of the brain) or brain damage secondary to the late pathophysiological processes.

An unexpected finding is the twice as small number of focal perfusion deficits per patient in women as compared with men.

Focal perfusion deficits were accompanied by diffuse regional hypoperfusion, as compared with cerebellar perfusion in most of the cerebral regions, excluding the temporal lobes. This may be the result of either loss of cerebral autoregulation (17, 18) or a diffuse early or late vasospasm occurring in up to 25% patients 2–7 days after the trauma (19). Brain oedema alone is possibly not an enhancing factor, as in our material no influence of speedy treatment introduction or treatment of the development of focal perfusion deficits was documented.

Of our findings the hypoperfusion of basal ganglia and thalami seems to be particularly worthy of attention. Basal ganglia lesion may explain such post-concussion symptoms as depression, behaviour problems, intellectual and emotional disorders (20, 21). The thalamic lesion may result in disturbed sleep, pain and motivation (22). The high incidence of focal thalamic lesions after head trauma, up to 47%, has been reported previously (10). Although one should be cautious in the interpretation of focal lesions in such small structures, which are susceptible to reangulation artefacts, our results seem to confirm their results, in part at least. The hypoperfusion of those structures could follow a vasospasm of basal intracranial arteries, mostly basilar and middle cerebral artery (18). The latter are frequent in cervical spine whiplash injury (13), therefore a thoroughly collected case history should accompany the referral for SPECT scanning.

## Practical conclusions

A practical matter is SPECT's superiority over CT scanning and a question of what this really means. This might be the result of SPECT's ability to show the incomplete infarcts (selective neuronal loss), i.e. loss of neurones without any breaking of the blood-brain-barrier and the development of focal necrosis (23). Alternatively, SPECT is able to show the ischaemic penumbra, which enables it to show the hypoperfused area of size theoretically below the spatial resolution of SPECT.

But what does all this mean in practice for the neurologist and/or traumatologist? SPECT cannot replace traditional structural imaging in detecting haematoma or oedema, its influence on the course of treatment of mild head trauma is also doubtful.

In the authors' opinion SPECT will play a major role in three cases:

- in the early period after trauma, documenting brain lesion in patients with likely judicial consequences in the future (victims of car accidents and criminal incidents);
- in detecting cases of simulation;
- in the remote period after trauma, assessing patients with post-concussion syndrome.

Referring to the particular points:

A victim of car accidents and criminal incidents will possibly make legal claims after discharge. Therefore, an objective assessment of the victim's health should be made in order to have a case history and documentation which are as complete as possible. SPECT scanning, a method much more sensitive than CT scanning, should be performed early, as a reference for future monitoring.

SPECT scanning is not entirely specific, but we have shown its normal pattern in patients whose cranio-cerebral trauma history is at least doubtful (for example, potential army conscripts). Normal SPECT scanning, at least in younger patients, could be an excluding factor in cases of simulation. Possibly in such cases SPECT cannot serve as the crowning proof alone, but it could be an important element.

The most important indication would be with patients with post-concussion syndrome. There is always a question whether post-concussion memory impairment, sleep and mood disturbances and poor concentration are the result of organic brain lesion or rather have psychogenic origin. Normal brain SPECT results could help in excluding organic brain lesion.

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